=> S CHROMOSOME (2A) (18) AND (BIPOLAR OR MANIC)

6999 CHROMOSOME 1649693 18

126 CHROMOSOME (2A) (18)

37700 BIPOLAR 414 MANIC

2 CHROMOSOME(2A)(18) AND (BIPOLAR OR MANIC)

=> D L1 1-2 CIT, AB

1. 5,914,394 Jun. 22, 1999 Methods and compositions for the diagnosis and treatment of neuropsychiatric disorders; Hong Chen, et al., 536/23.5; 435/69.1, 70.2, 91.1, 252.3, 320.1, 325, 333; 536/23.1, 24.1 [INAGE AVALLABLE]

US PAT NO: 5,914,394 [IMAGE AVAILABLE]

L1: 1 of 2

ABSTRACT:

1.1

The present invention relates to the mammalian fsh16 gene, a novel gene associated with bipolar affective disorder (BAD) in humans. The invention encompasses fsh16 nucleic acids, recombinant DNA molecules, cloned genes or degenerate variants thereof, fsh16 gene products and antibodies directed against such gene products, cloning vectors containing mammalian fsh16 gene molecules, and hosts that have been genetically engineered to express such molecules. The invention further relates to methods for the identification of compounds that modulate the expression of fshl6 and to using such compounds as therapeutic agents in the treatment of fshl6 disorders and neuropsychiatric disorders. The invention also relates to methods for the diagnostic evaluation, genetic testing and prognosis of fsh16 disorders and neuropsychiatric disorders including schizophrenia, attention deficit disorder, a schizoaffective disorder, a bipolar affective disorder or a unipolar affective disorder, and to methods and compositions for the treatment these disorders.

2. 5,866,412, Feb. 2, 1999, Chromosome 18 marker; Hong Chen, et al., 435/320.1, 243, 325; 536/23.1, 23.5 [IMAGE AVAILABLE]

US PAT NO: 5,866,412 [IMAGE AVAILABLE]

L1: '2 of 2

ABSTRACT:

ABSTRACT:
The present invention relates to the mammalian fshl5w6 gene, a novel gene associated with bipolar affective disorder (BAD) in humans. The invention encompasses fshl5w6 nucleic acids, recombinant DNA molecules, cloned genes or degenerate variants thereof, fshl5w6 gene products and antibodies directed against such gene products, cloning vectors containing mammalian fshl5w6 gene molecules, and hosts that have been genetically engineered to express such molecules. The invention further relates to methods for the identification of compounds that modulate the expression of fshl5w6 and to using such compounds as therapeutic agents in the treatment of fshl5w6 disorders and neuropsychiatric disorders. The invention also relates to methods for the diagnostic evaluation, genetic testing and prognosis of fshl5w6 disorders and neuropsychiatric disorders including schizophrenia, attention deficit disorder, a schizoaffective disorder, a hipolar affective disorder or a unipolar affective disorders, and to methods and compositions for the treatment these disorders.

- ANSWER 1 OF 6 MEDLINE
- MEDLINE 1999264248 AN
- DN
- Assessing the feasibility of linkage disequilibrium methods for mapping TI complex traits: an initial screen for bipolar disorder
- Escamilla M A; McInnes L A; Spesny M; Reus V I; Service S K; Shimayoshi loci on chromosome 18. AU
- Tyler D J; Silva S; Molina J; Gallegos A; Meza L; Cruz M L; Batki S; N: Vinogradov S; Neylan T; Nguyen J B; Fournier E; Araya C; Barondes S H; Leon P; Sandkuijl L A; Freimer N B
- Neurogenetics Laboratory, University of California San Francisco, San CS
- Francisco, USA. MH00916 (NIMH) NC
- MH49499 (NIMH) MH48695 (NIMH)
- AMERICAN JOURNAL OF HUMAN GENETICS, (1999 Jun) 64 (6) 1670-8. Journal code: 3IM. ISSN: 0002-9297.
- United States Journal; Article; (JOURNAL ARTICLE)
- English LA
- Priority Journals FS
- EM 199908
- 19990804 EW
- Linkage disequilibrium (LD) analysis has been promoted as a method of mapping disease genes, particularly in isolated populations, but has not yet been used for genome-screening studies of complex disorders. We present results of a study to investigate the feasibility of LD methods for genome screening using a sample of individuals affected with severe bipolar mood disorder (BP-I), from an isolated population of the Costa Rican central valley. Forty-eight patients with BP-I were genotyped for markers spaced at approximately 6-cM intervals across chromosome 18. Chromosome 18 was chosen because a previous genome-screening linkage study of two Costa
 - Rican families had suggested a BP-I locus on this chromosome. Results of the current study suggest that LD methods will be useful for mapping BP-I in a larger sample. The results also support previously reported possible localizations (obtained from a separate collection of patients) of BP-I-susceptibility genes at two distinct sites on this chromosome. Current limitations of LD screening for identifying loci for complex traits are discussed, and recommendations are made for future research with these methods.
- ANSWER 2 OF 6 MEDLINE L2
- 1998351040 MEDITNE AN
- 98351040 DN
- Affective disorder associated with a balanced translocation TI
- involving chromosome 14 and 18.
- Overhauser J; Berrettini W H; Rojas K ΑU
- Department of Biochemistry and Molecular Pharmacology, Thomas Jefferson University, Philadelphia, PA 19107, USA. J Overhauser@lac.jci.tju.edu PSYCHARTIC GENETICS, (1998 Summer) 8 (2) 53-6. CS
- SO Journal code: B3X. ISSN: 0955-8829.
- ENGLAND: United Kingdom CY
- Journal; Article; (JOURNAL ARTICLE) DT
- English LA
- Priority Journals FS
- EM 199901

We report a case of a women with psychiatric illness that includes ΕШ

bipolar disorder who has a karyotype of $\frac{1}{46}$, XX, t (14;18) (q11.2;q22.1). The region on chromosome 18 that is involved in the translocation has been implicated in other families through linkage and association studies as possibly containing a gene for bipolar illness.

ANSWER 3 OF 6 MEDLINE

MEDLINE 97480722 AN

DN

Genomic structure and chromosomal localization of a human myo-inositol TI monophosphatase gene (IMPA).

Sjoholt G; Molven A; Lovlie R; Wilcox A; Sikela J M; Steen V M

Dr. Einar Martens' Research Group for Biological Psychiatry, Center for AII CS Molecular Medicine, Haukeland University Hospital, Bergen, Norway.

GENOMICS, (1997 Oct 1) 45 (1) 113-22. 90 Journal code: GEN. ISSN: 0888-7543.

United States

Journal; Article; (JOURNAL ARTICLE) DT

English LA

Priority Journals GENBANK-Y11360; GENBANK-Y11361; GENBANK-Y11362; GENBANK-Y11363; FS OS GENBANK-Y11364; GENBANK-Y11365; GENBANK-Y11366; GENBANK-Y11367

199801 EΜ

19980104

EW Manic-depressive illness is a serious psychiatric disorder that in many, but far from all, patients can be treated with lithium. The main causes for discontinuation of lithium therapy are unpleasant or serious side effects and lack of response. The reason for the striking variation in clinical efficacy of lithium treatment among bipolar patients is not known. The enzyme myo-inositol monophosphatase (IMPase) has been postulated as a target for the mood-stabilizing effects of lithium, but variation in the coding region of the human IMPA gene encoding IMPase activity has not been observed in manic-depressive patients (Steen et al., Pharmacogenetics, 1996, 6, 113-116). It is nevertheless conceivable that polymorphisms or mutations in the noncoding regions of this gene could influence the lithium response in psychiatric patients. As a first step in investigating this possibility, we here report the genomic structure of the human IMPA gene. The gene is composed of at least nine exons and covers more than 20 kb of sequence on chromosome 8q21.13-q21.3. In the 3'-untranslated part of the gene, we observed a polymorphism (a G to A transition) and also two short

similar to the inositol/cholin-responsive element consensus. Finally, we postulate that two additional IMPA-like transcripts originate from the human genome, one from a position close to IMPA itself on chromosome 8

and the other from chromosome 18p. Our data may contribute to the identification of genetic factors involved in the pathogenesis and determination of treatment response in manic-depressive illness.

ANSWER 4 OF 6 MEDLINE MEDLINE

97430985 AN 97430985

DN Lack of evidence for a major locus for bipolar disorder in the pericentromeric region of chromosome 18 in

Irish pedigrees. Mynett-Johnson L A; Murphy V E; Manley P; Shields D C; McKeon P ΑU Department of Genetics, Trinity College Dublin, Ireland.

CS BIOLOGICAL PSYCHIATRY, (1997 Sep 15) 42 (6) 486-94. SO

Journal code: A3s. ISSN: 0006-3223.

United States CY

(CLINICAL TRIAL) DΨ Journal; Article; (JOURNAL ARTICLE)

English LA

Priority Journals FS

199712 EM

EW

Seven families, multiply affected by bipolar mood AB

disorder, have been collected from the Irish population and have been genotyped with microsatellite markers from the pericentromeric

of chromosome 18, a region that has been implicated as a site for a susceptibility gene for this relative common psychiatric disorder. The families significantly excluded linkage of

bipolar disorder to this region under various models. Although the data provided no evidence of linkage heterogeneity among families, the number of families investigated may be too small to exclude completely the possibility of linkage in a small number of families.

ANSWER 5 OF 6 MEDLINE

97372982 MEDLINE AN

DN 97372982

Cytogenetic abnormalities on chromosome 18 associated with bipolar affective disorder or schizophrenia. TI

Mors O; Ewald H; Blackwood D; Muir W DII

Institute for Basic Psychiatric Research, Risskov, Denmark. CS

BRITISH JOURNAL OF PSYCHIATRY, (1997 Mar) 170 278-80.

Journal code: BlK. ISSN: 0007-1250.

ENGLAND: United Kingdom CY

Journal; Article; (JOURNAL ARTICLE) DT

LΑ English

who

Priority Journals FS

EM

BACKGROUND: A few recent linkage studies have shown a possible locus for 199710

bipolar disorder on chromosome 18. Cytogenetic studies may assist in the further localisation of susceptibility loci on this chromosome. METHOD: A search was made for abnormalities of chromosome 18 in two separate large cytogenetic databases. In Denmark detection of mental illness in subjects with chromosome abnormalities was done by cross-linking the two separate register of psychiatric and chromosome disorders. In Scotland the Cytogenetic Registry of the MRC Human Genetics Unit undertakes long-term

clinical follow-up of all cases with chromosome abnormalities. RESULTS: Cross-linking the two Danish register's revealed a family with the rare karyotype abnormality inv(18) (p11.3;q21.1) with one inversion carrier

also suffered from bipolar disorder. In this family there were two other cases of bipolar disorder, but the karyotype of these cases could not be established. One family in Scotland showed a case of schizophrenia in a carrier of inv(18) with the same breakpoints as the Danish family. CONCLUSIONS: We suggest further studies of the 18p11.3 and 18q21.1 regions in order to identify genes involved in bipolar affective disorder and schizophrenia.

ANSWER 6 OF 6 MEDLINE T.2

97209418 MEDLINE MA

97209418 DN

Genetics of manic depressive illness. TΙ

MacKinnon D F; Jamison K R; DePaulo J R Department of Psychiatry, Johns Hopkins University School of Medicine, AU

Baltimore, Maryland 21287, USA. ANNUAL REVIEW OF NEUROSCIENCE, (1997) 20 355-73. Ref: 65

Journal code: 5Z5. ISSN: 0147-006X.

United States CY

Journal; Article; (JOURNAL ARTICLE) DΨ General Review; (REVIEW)

(REVIEW, TUTORIAL)

LA English

FS Priority Journals

EM 199708

AB Manic depressive illness is a common and frequently debilitating familial psychiatric disorder. Efforts to understand the mechanisms of inheritance have been hindered by the complexity of the phenotype, which may range from benign mood swings to chronic psychosis, and by apparently nonmendelian modes of transmission. Early reports of linkage

to

chromosomal loci have fallen into doubt; however they have helped encourage the development of more sophisticated methods for analyzing complex phenotypes. Using such methods, linkage of manic depressive illness to loci on chromosome 18 has been reported and apparently replicated, and work is proceeding to identify genes

associated

with what is probably a genetically heterogeneous set of disorders. As molecular mechanisms of inheritance are elucidated, it will be important to consider the ethical implications of genetic testing in a clinically and genetically complex disorder such as manic depressive illness.

- ANSWER 1 OF 4 MEDLINE L6 MEDLINE 1998019047 AN
- 98019047 DN
 - Linkage analysis of manic depression (bipolar
- affective disorder) in Icelandic and British kindreds using markers on
- the
- Kalsi G; Smyth C; Brynjolfsson J; Sherrington R S; O'Neill J; Curtis D; AU
- Rifkin L; Murphy P; Petursson H; Gurling H M Molecular Psychiatry Laboratory, University College London Medical CS
- School,
- HK ... HUMAN HEREDITY, (1997 Sep-Oct) 47 (5) 268-78. 50 Journal code: GE9. ISSN: 0001-5652.
- Switzerland
- Journal; Article; (JOURNAL ARTICLE)
- LA English
- Priority Journals FS
- EM 199802
- EW
- Attempts were made to follow up results of a previous linkage study which suggested that a locus-modifying susceptibility to bipolar and related
- unipolar affective disorder might be present in the pericentromeric
- of the short arm of chromosome 18. Twenty-three region
- multiply affected pedigrees collected from Iceland and the UK were genotyped using three highly polymorphic microsatellite markers at
- D18S37.
- D18540 and D18544 which span the region implicated. Lod score analyses under the assumption of heterogeneity and non-parametric linkage analyses were performed. The total lod scores obtained were strongly negative, and analysis allowing for heterogeneity did not suggest that any subgroup of the families was linked. Model-free linkage analysis using extended relative pair analysis and MFLINK also failed to detect any evidence for linkage. Our study provides no support for the presence of a
 - locus-modifying genetic susceptibility to bipolar affective disorder in the pericentromeric region of chromosome 18q11.
 - Further analyses in independent samples should help to reveal whether our negative results are due to locus heterogeneity or whether the original results were false-positive.
- ANSWER 2 OF 4 MEDLINE 1.6
- MEDLINE AN 96304711
- 96304711 DM
- Maternal inheritance and chromosome 18 allele sharing ΨT
- in unilineal bipolar illness pedigrees.
- Gershon E S; Badner J A; Detera-Wadleigh S D; Ferraro T N; Berrettini W H National Institute of Mental Health, Bethesda, Maryland 20892-1274, USA. AU CS
- NC
- AMERICAN JOURNAL OF MEDICAL GENETICS, (1996 Apr 9) 67 (2) 202-7. SO Journal code: 3L4. ISSN: 0148-7299.
- United States CY
- Journal; Article; (JOURNAL ARTICLE) DT
- English LA
- Priority Journals FS
- We have replicated the observation of McMahon et al. [1995] that there is EM excess maternal transmission of illness in a series of previously
 - described unilineal Bipolar manic-depressive illness

extended pedigrees [Berrettini et al., 1991]. ("Transmission" is defined for any ill person in a pedigree when father or mother has a personal or immediate family history of major affective disorder.) We divided our pedigrees into exclusively maternal transmission (Mat) and mixed matérnal-paternal transmission (in different pedigree branches) (Pat). Using affected sib-pair-analysis, linkage to a series of markers on chromosome 18p-cen was observed in the Pat but not the Mat pedigrees, with significantly greater identity by descent (IBD) at these markers in the Pat pedigrees. As compared with the pedigree series as a whole, the proportion of alleles IBD in the linkage region is much increased in the Pat pedigrees. As shown by Kruglyak and Lander [1995],

- 8.8 the sharing proportion of alleles in affected relative pairs increases, the number of such pairs needed to resolve the linkage region to a 1 cM interval becomes smaller. Genetic subdivision of an illness by clinical
- or pedigree configuration criteria may thus play an important role in discovery of disease susceptibility mutations.
- ANSWER 3 OF 4 MEDLINE
- 96301288 ΔM MEDIATNE
- 96301288 DM
- TТ Analysis of chromosome 18 DNA markers in multiplex
- pedigrees with manic depression.
- 2511 Coon H; Hoff M; Holik J; Hadley D; Fang N; Reimherr F; Wender P; Byerley
- CS Department of Psychiatry, University of Utah Medical School, Salt Lake City 84121, USA.
- MH-44212 (NIMH) NC:
 - MH10168-F32 (NIMH) MO1-RR00064 (NCRR)
- BIOLOGICAL PSYCHIATRY, (1996 Apr 15) 39 (8) 689-96. SO
 - Journal code: A3S. ISSN: 0006-3223. United States
- Journal; Article; (JOURNAL ARTICLE)
- LA English
- FS Priority Journals 199612
- EM
- AB Six pedigrees segregating manic-depressive illness
 - (MDI) were analyzed for linkage to 21 highly polymorphic microsatellite DNA markers on chromosome 18. These markers span almost the entire length of the chromosome, and gaps between markers are

less than 20 cM. In particular, we analyzed several markers localizing to the pericentromeric region of chromosome 18 which generated lod scores suggestive of linkage in an independent study. Lod

score analysis was performed and results were examined by family. One region produced positive lod scores, though at 18q23 and not in the pericentromeric region. We additionally used two nonparametric methods

because the true mode of transmission of MDI is unknown; results were again somewhat suggestive for markers in the region of 18q23 but not in the pericentromeric region.

- 1.6 ANSWER 4 OF 4 MEDLINE
- 94286549 MEDLINE AN
- DM 94286549
- TI Chromosome 18 DNA markers and manic-
- depressive illness: evidence for a susceptibility gene.
- Alt Berrettini W H; Ferraro T N; Goldin L R; Weeks D E; Detera-Wadleigh S; Nurnberger J I Jr; Gershon E S
- Department of Psychiatry and Human Behavior, Jefferson Medical College, Thomas Jefferson University, Philadelphia, PA 19107.
- MC 1 P41 RR03655 (NCRR)
- 30 PROCEEDINGS OF THE NATIONAL ACADEMY OF SCIENCES OF THE UNITED STATES OF

AMERICA, (1994 Jun 21) 91 (13) 5918-21. Journal code: PV3. ISSN: 0027-8424. United States

CY DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals; Cancer Journals

199409 EM

In the course of a systematic genomic survey, 22 manic-

depressive (bipolar) families were examined for linkage to 11 chromosome 18 pericentromeric marker loci, under dominant and recessive models. Overall logarithm of odds score analysis for the pedigree series was not significant under either model, but several families yielded logarithm of odds scores consistent with linkage under dominant or recessive models. Affected sibling pair analysis of these data yielded evidence for linkage (P < 0.001) at D18S21. Affected pedigree member analysis also suggests linkage, with multilocus results for five loci giving P < 0.0001 and P = 0.0007 for weighting functions f(p) = 1 and 1/square root p, respectively, where p is the allele frequency. These results imply a susceptibility gene in the pericentromeric region of chromosome 18, with a complex mode of inheritance. Two plausible candidate genes, a corticotropin receptor and the alpha subunit of a GTP binding protein, have been localized to this region.

=> d his

(FILE 'HOME' ENTERED AT 14:29:09 ON 13 JUL 1999)

FILE 'MEDLINE' ENTERED AT 14:29:25 ON 13 JUL 1999

1488 S BIPOLAR AND MOOD AND DISORDER L1 L2 6 S L1 AND CHROMOSOME (2A) (18?)

L3 153 S MANIC AND DEPRESS? AND (18?)

L.4 1655 S CHROMOSOME (4A) (18?)

L5 6 S L4 AND L3 1.6

4 S L5 NOT L2

=> s 18 and (bipolar or manic)

18536 BIPOLAR 1039 MANIC

21 L8 AND (BIPOLAR OR MANIC)

=> d 19 1-21 bib, ab

ANSWER 1 OF 21 CA COPYRIGHT 1999 ACS

131:1252 CA AN

TT CCG repeats in cDNAs from human brain

Kleiderlein, John J.; Nisson, Paul E.; Jessee, Joel; Li, W.-B.; Becker,

G.; Derby, Michael L.; Ross, Christopher A.; Margolis, R. L. Department of Psychiatry, Division of Neurobiology, The Laboratories of Genetic Neurobiology and Molecular Neurobiology, Johns Hopkins University School of Medicine, Baltimore, MD, 21287, USA

90 Hum. Genet. (1998), 103(6), 666-673 CODEN: HUGEDQ; ISSN: 0340-6717

PB Springer-Verlag

DT Journal

LA English

AB Expansion mutations of trinucleotide repeats and other units of unstable DNA have been proposed to account for at least some of the genetic susceptibility to a no. of neuropsychiatric disorders, including bipolar affective disorder, schizophrenia, autism, and panic disorder. To generate addnl. candidate genes for these and other disorders, cDNA libraries from human brain were probed at high stringency for clones contg. CCG, CGC, GCC, CGG, GCG, and GGC repeats (referred to collectively as CCG repeats). Some 18 cDNAs contg. previously

unpublished or uncharacterized repeats were characterized for chromosomal locus, repeat length polymorphism, and similarity to genes of known function. The cDNAs were also compared with the 37 human genes with eight or more consecutive CCG triplets in GenBank. The repeats were mapped to a no. of loci, including 1p34, 2p11.2, 2q30-32, 3p21, 3p22, 4q35, 6q22, 7qter, 13p13, 17q24, (Bp11) 19p13.3, 20q12, 20q13.3, and 22q12. Length polymorphism was detected in 50% of the repeats. The newly cloned cDNAs include a complete transcript of human neurexin-1B, a portion of BCNG-1

- (a newly described brain-specific ion channel), a previously unreported polymorphic repeat located in the 5' UTR region of the guanine nucleotide-binding protein (G-protein) .beta.2 subunit, and a human version of the mouse proline-rich protein 7. This list of cDNAs should expedite the search for expansion mutations assocd. With diseases of the central nervous system.
- ANSWER 2 OF 21 CA COPYRIGHT 1999 ACS

AN 130:178369 CA

ZGGBP1 proteins related to bipolar affective disorder type 1 TΤ

Flannery, Angela Veronica; Finnegan, Maria Christina Martina IN PA

Zeneca Limited, UK

PCT Int. Appl., 58 pp. SO CODEN: PIXXD2

DT Patent

LA English

FAN.CNT 1

PATENT NO.

KIND DATE

APPLICATION NO. DATE

19980728 A1 19990211 WO 98-GB2259 PT WO 9906539 W: JP, US

RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE

19970801 PRAI GB 97-16162

AB A new human gene (ZGGBP1) is described which is assocd. with neurol. affective disorders such as bipolar affective disorder. A full-length cDNA encoding human ZGGBP1 and a partial cDNA encoding murine ZGGBP1 are disclosed. Polymorphic variants of the gene and functional domains encoded within the gene are also provided. The gene maps to

human

chromosome 18q21 and shows apprecrable sequence homol. to the ned-4 gene on chromosome 15. The invention further relates to methods for identifying compds. which modulate the activity of ZGGBP1 protein, and to diagnostic assays for the detection of ZGGBP1 in biol. samples.

ANSWER 3 OF 21 CA COPYRIGHT 1999 ACS L9

AN 130:172974 CA

Use of fsh05 gene and protein for the diagnosis and treatment of TT neuropsychiatric disorders

TN Chen, Hong; Freimer, Nelson B.

Millennium Pharmaceuticals, Inc., USA; The Regents of the University of PA California

PCT Int. Appl., 117 pp. 30 CODEN: PIXXD2

DT Patent

LA English

FAN. CNT 1

APPLICATION NO. DATE PATENT NO. KIND DATE ----9904825 A1 19990204 Wo 98-US15183 19980722 WO 9904825 PT W: AU, CA, JP RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE AU 98-85805 AU 9885805 A1 19990216 19980722

PRAI US 97-898082 19970722 19980722 WO 98-US15183

- The present invention relates to the mammalian fsh05 gene, a novel gene assocd. with bipolar affective disorder (BAD) in humans. The invention encompasses fsh05 nucleic acids, recombinant DNA mols., cloned genes or degenerate variants ther eof, fsh05 gene products and antibodies directed against such gene products, cloning vectors contg. mammalian fsh05 gene mols., and hosts that have been genetically engineered to express such mols. The invention further relates to methods for the identification of compds. that modulate the expression of fsh05 and to using such compds. as therapeutic agents in the treatment of fsh05 disorders and neuropsychiatric disorders. The invention also relates to methods for the diagnostic evaluation, genetic testing and prognosis of fsh05 disorders and neuropsychiatric disorders including schizophrenia, attention deficit disorder, a schizoaffective disorder, a bipolar affective disorder or a unipolar affective disorder, and to methods and compns. for the treatment of these disorders.
- ANSWER 4 OF 21 CA COPYRIGHT 1999 ACS L9

AN 129:340353 CA

No evidence for significant linkage between bipolar affective disorder and chromosome 18 pericentromeric markers in a large series of multiplex extended pedigrees

Knowles, James A.; Rao, Peter A.; Cox-Matise, Tara; Loth, Jo Ellen; De AU vesus, Gracielle M.; Levine, Laura; Das, Kamna; Penchaszadeh, Graciela

K.; Alexander, Joyce R.; Lerer, Bernard; Endicott, Jean; Ott, Jurg; Gilliam, T. Conrad; Baron, Miron

Columbia University College of Physicians and Surgeons and New York State CS

- Psychiatric Institute, Rockefeller University, New York, NY, 10032, USA Am. J. Hum. Genet. (1998), 62(4), 916-924 CODEN: AJHGAG; ISSN: 0002-9297

 PB University of Chicago Press
 DT Journal
 LA English
 Bhpolar affective disorder (BP) is a major neuropsychiatric
- AB Bapolar affective disorder (BP) is a major neuropsychiatric disorder with high heritability and complex inheritance. Previously reported linkage between BP and DNA markers in the pericentromeric region of chromosome 18, with a parent-of-origin effect (linkage was present in pedigrees with paternal transmission and absent in
 - pedigrees with exclusive maternal inheritance), has been a focus of interest in human genetics. We reexamd, the evidence in one of the largest samples reported to date (1013 genotyped individuals in 53 unilineal multiplex pedigrees), using 10 highly polymorphic markers and a range of parametric and nonparametric analyses. There was no evidence
- significant linkage between BF and chromosome 18
 pericentromeric markers in the sample as a whole, nor was there evidence
 for significant parent-of-origin effect (pedigrees with paternal
 transmission were not differentially linked to the implicated chromosomal
 region). Two-point LOD scores and single-locus sib-pair results gave
 - support for suggestive linkage, but this was not substantiated by multilocus anal., and the results were further tempered by multiple test effects. We conclude that there is no compelling evidence for linkage between BP and chromosome 18 pericentromeric markers in this sample.
- L9 ANSWER 5 OF 21 CA COPYRIGHT 1999 ACS
- AN 129:287565 CA
- TI Methods and compositions for the diagnosis and treatment of
 - neuropsychiatric disorders N Chen, Hong; Freimer, Nelson B.
- IN Chen, Hong; Freimer, Nelson B.

 PA Millenium Pharmaceuticals, Inc., USA; The Regents of the University of
- California SO PCT Int. Appl., 94 pp.
- CODEN: PIXXD2

WO 98-US6208

DT Patent

for

- LA English
- EDM CMT 1

E MIA .	CNII				
	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
				~	
PI	WO 9842362	A1	19981001	WO 98-US6208	19980327
	W: AU, CA,	JP			

RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT,

AU 9867865 A1 19981020 AU 98-67865 19980327 PRAI US 97-828010 19970327

19980327

AB The present invention relates to the mammalian fsh05 gene, a novel gene assocd with bipolar affective disorder (BAD) in humans. The invention encompasses fsh05 nucleic acids, recombinant DNA mols., cloned genes or degenerate variants thereof, fsh05 gene products and antibodies directed against such gene products, cloning vectors contg. mammalian fsh05 gene mols., and hosts that have been genetically engineered to express such mols. The invention further relates to methods for the identification of compds. that modulate the expression of fsh05 and to using such compds. as therapeutic agents in the treatment of fsh05 disorders and neuropsychiatric disorders. The invention also relates to methods for the diagnostic evaluation, genetic testing and prognosis of fsh05 disorders and neuropsychiatric disorders including schizophrenia, attention deficit disorder, a schizoaffective disorder, a bipolar affective disorder, and to methods and

compns. for the treatment of these disorders.

- L9 ANSWER 6 OF 21 CA COPYRIGHT 1999 ACS
- AN 129:286742 CA
- TI Fsh16 gene and methods and compositions for the diagnosis and treatment of
- neuropsychiatric disorders
- IN Chen, Hong; Freimer, Nelson B.
- PA Millenium Pharmaceuticals, Inc., USA; The Regents of the University of California
- so PCT Int. Appl., 93 pp.
- CODEN: PIXXD2
- DT Patent
- LA English FAN.CNT 1

P

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9842726	A1	19981001	WO 98-US6210	19980327
	W: AU, CA,	JP			

RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE
US 5914394 A 19990622 US 97-828009 19970327

AU 9867867 A1 19981020 PRAI US 97-828009 19970327

WO 98-US6210 19980327 The present invention relates to the mammalian fshl6 gene, a novel gene AB assocd, with bipolar affective disorder (BAD) in humans. The invention encompasses fsh16 nucleic acids, recombinant DNA mols., cloned genes or degenerate variants thereof, fshl6 gene products and antibodies directed against such gene products, cloning vectors contg. mammalian fsh16 gene mols., and hosts that have been genetically engineered to express such mols. The invention further relates to methods for the identification of compds. that modulate the expression of fsh16 and to using such compds. as therapeutic agents in the treatment of fsh16 disorders and neuropsychiatric disorders. The invention also relates to methods for the diagnostic evaluation, genetic testing and prognosis of fsh16 disorders and neuropsychiatric disorders including schizophrenia, attention deficit disorder, a schizoaffective disorder, a bipolar affective disorder or a unipolar affective disorder, and to methods and

AU 98-67867

19980327

L9 ANSWER 7 OF 21 CA COPYRIGHT 1999 ACS

compns. for the treatment of these disorders.

- AN 129:286740 CA
- TI Fsh22 gene and methods and compositions for the diagnosis and treatment
- neuropsychiatric disorders
- IN Chen, Hong; Freimer, Nelson B.
- PA Millenium Pharmaceuticals, Inc., USA; The Regents of the University of California
- SO PCT Int. Appl., 93 pp.
- CODEN: PIXXD2 DT Patent
- T.A English
- FAN. CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9842723	A1	19981001	WO 98-US6209	19980327

RW: AT. BE, CH. DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT,

SE AU 9867866 A1 19981020 AU 98-67866 19980327

PRAI US 97-828008 19970327 WO 98-US6209 19980327

AB The present invention relates to the mammalian fsh22 gene, a novel gene assocd. with bipolar affective disorder (BAD) in humans. The

invention encompasses fsh22 nucleic acids, recombinant DNA mols., cloned genes or degenerate variants thereof, fsh22 gene products and antibodies directed against such gene products, cloning vectors contg. mammalian fsh22 gene mols., and hosts that have been genetically engineered to express such mols. The invention further relates to methods for the identification of compds. that modulate the expression of fsh22 and to using such compds. as therapeutic agents in the treatment of fsh22 disorders and neuropsychiatric disorders. The invention also relates to methods for the diagnostic evaluation, genetic testing and prognosis of fsh22 disorders and neuropsychiatric disorders including schizophrenia. attention deficit disorder, a schizoaffective disorder, a bipolar affective disorder or a unipolar affective disorder, and to methods and compns, for the treatment of these disorders,

- ANSWER 8 OF 21 CA COPYRIGHT 1999 ACS L9
- AN 129:271555 CA
- Fsh15w6 gene and methods and compositions for the diagnosis and treatment TT of neuropsychiatric disorders
- TN Chen, Hong; Freimer, Nelson B.
- PA Millenium Pharmaceuticals, Inc., USA; The Regents of the University of California
- PCT Int. Appl., 94 pp. SO
- CODEN: PIXXD2
- DT Patent
- T.Z. English
- FAN. CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9842724	A1	19981001	WO 98-US6211	19980327
	W: AU, CA,	JP			

- RW: AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE US 5866412 19990202 US 97-828007 A 19970327
- AU 9867868 Α1 19981020
- AU 98-67868 19980327 PRAI US 97-828007 19970327 WO 98-US6211 19980327
- The present invention relates to the mammalian fsh15w6 gene, a novel gene assocd, with bipolar affective disorder (BAD) in humans. The invention encompasses fsh15w6 nucleic acids, recombinant DNA mols.,
- cloned genes or degenerate variants thereof, fsh15w6 gene products and antibodies

directed against such gene products, cloning vectors contg. mammalian fsh15w6 gene mols., and hosts that have been genetically engineered to express such mols. The invention further relates to methods for the identification of compds. that modulate the expression of fsh15w6 and to using such compds. as therapeutic agents in the treatment of fsh15w6 disorders and neuropsychiatric disorders. The invention also relates to methods for the diagnostic evaluation, genetic testing and prognosis of fshl5w6 disorders and neuropsychiatric disorders including schizophrenia, attention deficit disorder, a schizoaffective disorder, a bipolar affective disorder or a unipolar affective disorder, and to methods and compns, for the treatment of these disorders.

- L9 ANSWER 9 OF 21 CA COPYRIGHT 1999 ACS
- 129:1406 CA
- TI Chromosomal markers and diagnostic tests for manic-depressive illness
- TN Detera-Wadleigh, Sevilla D.; Gershon, Elliot S.; Badner, Judith A.; Goldin, Lynn R.; Berrettini, Wade H.; Yoshikawa, Takeo; Sanders, Alan R.; Esterling, Lisa E.
- PA United States Dept. of Health and Human Services, USA; Detera-Wadleigh, Sevilla D.; Gershon, Elliot S.; Badner, Judith A.; Goldin, Lynn R.; Berrettini, Wade H.; Yoshikawa, Takeo; Sanders, Alan R.; Esterling, Lisa

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SO
     PCT Int. Appl., 119 pp.
     CODEN: PIXXD2
DT
     Patent
T.Z
   English
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FAN. CNT 1 PATENT NO. KIND DATE

~----WO 9818963 19980507 A1 Wo 97-US19381 19971028 W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE, DK, EE, ES, FI, GB, GE, GH, HU, ID, IL, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, US, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, ML, MR, NE, SN, TD, TG

APPLICATION NO. DATE

CA 2241855 AA 19980507 CA 97-2241855 19971028 AU 9851509 A1 19980522 AU 98-51509 19971028 PRAI US 96-29278 19961028

WO 97-US19381 19971028

Methods and compns. are provided for detg. a genotype assocd. with increased susceptibility to manic-depressive illness. The genotype is detd. using markers for a region of chromosome 18 exhibiting linkage disequil. with manic-depressive illness. The invention also provides for a novel myo-inositol monophosphatase protein encoded for on chromosome 18. Using direct cDNA selection and phys. mapping by PCR, 25 novel, chromosome 18-specific cDNAs expressed in infant brain have been identified and positionally cataloged. A cDNA for a gene assocd. With manic-depression was identified. Based on sequence homol. and presence of protein motifs, the gene is proposed to encode myo-inositol monophosphatase. The promoter region of the gene was also isolated and sequenced.

ANSWER 10 OF 21 CA COPYRIGHT 1999 ACS 1.9

 ΔN 128:253385 CA

TΤ Genomic screening in manic-depressive disorder

ΑU Verheyen, Geert R.; Van Broeckhoven, Christine

CS Laboratory of Neurogenetics, Flanders Interuniversity Institute for Biotechnology (VIB), Department of Biochemistry, Born-Bunge Foundation (BBS), University of Antwerp (UIA), Antwerp, B-2610, Belg. Wenner-Gren Int. Ser. (1998), 69(Genetics and Psychiatric Disorders), SO

147-163

CODEN: WGISEA; ISSN: 1356-0409

PB Elsevier Science Ltd.

DT Journal; General Review LA English

AB A review with .apprx.50 refs., providing an overview of the results of

the linkage studies in several bipolar disorder families performed in the authors' lab. The authors have mostly found neg. linkage results. However, Xq27-q28 could not be excluded and small, pos. LOD scores are obtained. Suggestive LOD scores were also found for linkage to 18q22.3-q23.

1.9 ANSWER 11 OF 21 CA COPYRIGHT 1999 ACS

AN

128:176630 CA TI Rapid cloning of expanded trinucleotide repeat sequences from genomic DNA Koob, Michael D.; Benzow, Kellie A.; Bird, Thomas D.; Day, John W.; ΑU

Moseley, Melinda L.; Ranum, Laura P. W. Dep. Neurol., Univ. Minnesota, Minneapolis, MN, 55455, USA CS

SO Nat. Genet. (1998), 18(1), 72-75 CODEN: NGENEC; ISSN: 1061-4036

PB Nature America

Journal DYP

LA English

AB Trinucleotide repeat expansions have been shown to cause a no. of neurodegenerative diseases. A hallmark of most of these diseases is the presence of anticipation, a decrease in the age at onset in consecutive generations due to the tendency of the unstable trinucleotide repeat to lengthen when passed from one generation to the next. The involvement of trinucleotide repeat expansions in a no. of other diseases - including familial spastic paraplegia, schizophrenia, hipolar affective disorder and spinocerebellar ataxia type 7 (SCA7) - is suggested both by

the presence of anticipation and by repeat expansion detection (RED)

anal.

of genomic DNA samples. The involvement of trinucleotide expansions in these diseases, however, can be conclusively confirmed only by the isolation of the expansions present in these populations and detailed anal. to assess each expansion as a possible pathogenic mutation. We describe a novel procedure for quick isolation of expanded trinucleotide repeats and the corresponding flanking nucleotide sequence directly from small amts. of genomic DNA by a process of Repeat Anal., Pooler Isolation and Detection of individual clones contg. expanded trinucleotide repeats (RAFID cloning). We have used this technique to clone the pathogenic

SCA7

 ${\tt CAG}$ expansion from an archived DNA sample of an individual affected with ataxia and retinal degeneration.

L9 ANSWER 12 OF 21 CA COPYRIGHT 1999 ACS

AN 128:10756 CA

- TI Genomic structure and chromosomal localization of a human myo-inositol monophosphatase gene (IMPA)
- AU Sjoholt, Gry; Molven, Anders; Lovlie, Roger; Wilcox, Andrea; Sikela, James

M.; Steen, Vidar M.

CS Dr. Einar Martens' Research Group for Bioogical Psychiatry, Center for Molecular Medicine, Haukeland University Hospital, Bergen, N-5021, Norway SO Genomics (1997), 45(1), 113-122

GO Genomics (1997), 45(1), 113-122 CODEN: GNMCEP; ISSN: 0888-7543

PB Academic

DT Journal

LA English

B Manic-depressive illness is a serious psychiatric disorder that in many, but far from all, patients can be treated with lithium. The

main

causes for discontinuation of lithium therapy are unpleasant or serious side effects and lack of response. The reason for the striking variation in clin. efficacy of lithium treatment among bipolar patients is not known. The enzyme myo-inositol monophosphatase (IMPase) has been postulated as a target for the mood-stabilizing effects of lithium, but variation in the coding region of the human IMPA gene encoding IMPase activity has not been obsd. In manic-depressive patients (Steen et al., Pharmacogenetics, 1996, 6, 113-116). It is nevertheless conceivable that polymorphisms or mutations in the noncoding regions of this gene could influence the lithium response in psychiatric patients. As a first step in investigating this possibility, we here report the genomic structure of the human IMPA gene. The gene is composed of at least nine exons and covers more than 20 kb of sequence on chromosome 8q21.13-q21.3. In the 3'-untranslated part of the gene, we obsd. a polymorphism (a § to A translation) and also two short sequences similar

to

the inositol/cholin-responsive element consensus. Finally, we postulate that two addnl. IMPA-like transcripts originate from the human genome,

one

from a position close to IMPA itself on chromosome 8 and the other from chromosome 18p. Our data may contribute to the identification of genetic factors involved in the pathogenesis and detn. of treatment response in manic-depressive illness.

- L9 ANSWER 13 OF 21 CA COPYRIGHT 1999 ACS
- AN 128:842 CA
- TI A novel, heritable, expanding CTG repeat in an intron of the SEF2-1 gene on chromosome 18g21.1
- AU Breschel, T. S.; McInnis, M. G.; Margolis, R. L.; Sirugo, G.;
- Corneliussen, B.; Simpson, S. G.; McMahon, F. J.; MacKinnon, D. F.; Xu,
- F.; Pleasant, N.; Huo, Y.; Ashworth, R. G.; Grundstrom, C.; Grundstrom, T.; Kidd, K. K.; DePaulo, J. R.; Ross, C. A.
- CS George Browne Genet. Lab., Dep. Esychiatry Behav. Sci., Johns Hopkins Univ. Sch. Med., Baltimore, MD, USA
- SO Hum. Mol. Genet. (1997), 6(11), 1855-1863 CODEN: HMGEE5; ISSN: 0964-6906
- PB Oxford University Press
- DT Journal
- LA English
- AB There are currently 13 diseases known to be caused by unstable triplet repeat mutations; however, there are some instances (as with FRAXF and FRA16) when these mutations appear to be asymptomatic. In a search for polymorphic CTG repeats as candidate genes for bipolar disorder, we screened a genomic human chromosome 18-specific library and identified a 1.6 kb clone (7,6A) with a CTG24 repeat that
- maps
 - to 18q21.1. The CTG repeat locus, termed CTG18.1, is located within an intron of human SEF2-1, a gene encoding a basic helix-loop-helix DNA binding protein involved in transcriptional regulation. The CTGn repeat is highly polymorphic and very enlarged alleles, consistent with expansions of up to CTG2100, were identified. PCR and Southern blot
- anal.
 - in pedigrees ascertained for a Johns Hopkins University bipolar disorder linkage study and in CEPH ref. pedigrees revealed a tripartite distribution of CTG[8.1 alleles with stable alleles (CTG10-CTG37), moderately enlarged and unstable alleles (CTG53-CTG250), and very enlarged, unstable alleles (CTG600-CTG2100). Moderately enlarged alleles were not assocd. with an abnormal phenotype and have a combined enlarged allele frequency of 3% in the CEPH and bipolar populations.

 Very enlarged alleles, detectable only by Southern blot anal. of genomic digests, have thus far been found in only three individuals from our bipolar pedigrees, and to date, have not been found in any of the CEPH ref. pedigrees. These enlarged alleles may arise, at least in part, via somatic mutation.
- L9 ANSWER 14 OF 21 CA COPYRIGHT 1999 ACS
- AN 127:327441 CA
- TI Methods for detecting bipolar mood disorder susceptibility locus on human chromosome (18q)
- IN Friemer, Nelson B.; Deon, Pedro; Reus, Victor I.; Sandkuijl, Lodewijk A.; Barondes, Samuel H.
- PA Regents of the University of California, USA; Friemer, Nelson B.; Leon, Pedro; Reus, Victor I.; Sandkuijl, Lodewijk A.; Barondes, Samuel H.
- SO PCT Int. Appl., 51 pp.
- CODEN: PIXXD2
- DT Patent
- LA English FAN.CNT 1

PATENT NO.	KIND	DATE	APPLICATION NO. DATE	
PI WO 9737043	Al	19971009	WO 97-US4904 19970327	
W: AL; AM,	AT, AU,	, AZ, BA, :	BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE,	
DK, EE,	ES, FI	, GB, GE,	GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ,	
LC, LK,	LR, LS	LT, LU,	LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL,	
PT, RO,	RU, SD	, SE, SG, :	SI, SK, TJ, TM, TR, TT, UA, UG, US, UZ,	
VN, YU,	AM, AZ	, BY, KG,	KZ, MD, RU, TJ, TM	
RW: GH, KE,	LS, MW.	, SD, SZ,	UG, AT, BE, CH, DE, DK, ES, FI, FR, GB,	
GR, IE,	IT, LU.	MC, NL,	PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN,	

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ML, MR, NE, SN, TD, TG
                                              CA 97-2247996
                                                                 19970327
                       AA
                              19971009
     CA 2247996
                                              AU 97-24238
                                                                 19970327
                              19971022
     AU 9724238
                        A1
                                              WO 97-US14892
                                                                 19970822
                              19980226
     WO 9807887
                        A1
         W: AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CU, CZ, DE,
             DK, EE, ES, FI, GB, GE, GH, HU, IL, IS, JP, KE, KG, KP, KR, KZ,
              LC, LK, LR, LS, LT, LU, LV, MD, MG, MK, MN, MW, MX, NO, NZ, PL,
             PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, UA, UG, UZ, VN, YU, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM
         RW: GH, KE, LS, MW, SD, SZ, UG, ZW, AT, BE, CH, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA,
              GN, ML, MR, NE, SN, TD, TG
                                              AII 97-41604
                                                                 19970822
     AU 9741604
                       Al 19980306
PRAI US 96-14498
                        19960329
                       19960823
     US 96-23438
     WO 97-US4904
                       19970327
                       19970822
     WO 97-US14892
     The present invention is directed to methods of detecting the presence of
ΔB
     a bipolar mood disorder susceptibility locus in an individual,
     comprising analyzing a sample of DNA for the presence of a DNA
     polymorphism on the long arm of chromosome 18 between
     markers D18S469 and D18S554, wherein the DNA polymorphism is assocd. With
     a form of bipolar mood disorder (BP). The invention for the
     first time provides strong evidence of a susceptibility gene for BP that
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X

is located in the 18g22-q23 region of the long arm of chromosome 18. The disclosure describes the use of linkage anal, and genetic markers in the 18g22-q23 region to fine map the region and the use of genetic markers to genetically diagnose (genotype) BP in individuals, to confirm phenotypic diagnoses of BP, to det. appropriate treatments for patients with particular genotypic subtypes. Isolated polynuclectides useful for genetic linkage anal. of BP-I and methods for obtaining such isolated polynuclectides are also described. In screening for a BP susceptibility locus, only those individuals with the most severe and clin. distinctive form of BP were considered as affected. Two large pedigrees were selected from a genetically homogeneous population, that

the Central Valley of Costa Rica. The entire human genome was screened for linkage using mapped microsatellite markers and a model for genetic anal. in which most of the linkage information derived from affected individuals. Three lines of evidence supported the localization of a BP susceptibility locus to 18422-923: assocn. anal., linkage anal., and direct observation of a conserved marker haplotype.

9 ANSWER 15 OF 21 CA COPYRIGHT 1999 ACS

AN 126:5800 CA

A complete genome screen for genes predisposing to severe bipolar disorder in two Costa Rican pedigrees

AU McInnes, L. Alison; Escamilla, Michael A.; Service, Susan K.; Reus,

Victor I.; Leon, Pedro; Silva, Sandra; Rojas, Eugenia; Spesny, Mitzi; Baharloo, Siamak; et al.

CS Neurogenet. Lab., Univ. California, San Francisco, CA, 94143, USA

SO Proc. Natl. Acad. Sci. U. S. A. (1996), 93(23), 13060-13065

CODEN: PNASA6; ISSN: 0027-8424 National Academy of Sciences

PB National Acad DT Journal

LA English

English

Bipolar mood disorder (BP) is a debilitating syndrome characterized by episodes of mania and depression. We designed a multistage study to detect all major loci predisposing to severe BP (termed BP-I) in two pedigrees drawn from the Central Valley of Costa Rica, where the population is largely descended from a few founders in

the 16th-18th centuries. We considered only individuals with BP-I as affected

and screened the genome for linkage with 473 microsatellite markers. We used a model for linkage anal, that incorporated a high phenocopy rate

and

a conservative est. of penetrance. Our goal in this study was not to establish definitive linkage but rather to detect all regions possibly harboring major genes for BP-I in these pedigrees. To facilitate this aim, we evaluated the degree to which markers that were informative in

our

data set provided coverage of each genome region, we est. that at least 94% of the genome has been covered, at a predesignated threshold detd. through prior linkage simulation analyses. We report here the results of our genome screen for BP-I loci and indicate several regions that merit further study, including segments in 18q, 18p, and 11p, in which suggestive lod scores were obsd. for two or more contiguous markers. Isolated lod scores that exceeded our thresholds in one or both families also occurred on chromosomes 1, 2, 3, 4, 5, 7, 13, 15, 16, and 17. Interesting regions highlighted in this genome screen will be followed up using linkage disequil (LD) methods.

L9 ANSWER 16 OF 21 CA COPYRIGHT 1999 ACS

AN 125:192566 CA

TI No association between chromosome-18 markers and lithium-responsive affective disorders

AU Turecki, Gustavo: Alda, Martin; Grof, Paul; Grof, Eva; Martin, Rory; Cavazzoni, Patrizia A.; Duffy, Anne; Maciel, Patricia; Rouleau, Guy A. Centre Research Neuroscience, Montreal General Hospital, Montreal, PQ,

нзн

Psychiatry Res. (1996), 63(1), 17-23

CODEN: PSRSDR; ISSN: 0165-1781 DT Journal

so

LA English

BA An allelic assocn. study of excellent responders to lithium was conducted with a candidate gene (Golf, a G-protein receptor gene) and five other chromosome-18p markers. Golf is of special interest because it maps to a region of chromosome 18 where two independent groups (Berrettini et al., 1994; Stine et al., 1995) have found linkage to bipolar disorder. It has been proposed that G proteins are involved in the pathogenessis of bipolar disorder, and lithium, an effective prophylactic agent, is known to impair G-protein activation. To reduce heterogeneity - a common obstacle to genetic investigation - only patients who showed excellent response to lithium prophylaxis were studied. Fifty-five qenetically unrelated excellent responder responders.

lithium

prophylaxis were compared with 94 normal subjects of similar ethnic background. The groups did not differ in either allele or genotype frequency for the tested markers. The data do not support the hypothesis that the tested loci confer a major susceptibility for affective disorders.

L9 ANSWER 17 OF 21 CA COPYRIGHT 1999 ACS

AN 125:2666 CA

TI Genetic mapping using haplotype, association and linkage methods suggests

a locus for severe bipolar disorder (BPI) at 18q22-q23

AU Freimer, Nelson B.; Reus, Victor I.; Escamilla Michael A.; McInnes, L. Alison; Spesny, Mitzi: Leon, Pedro; Service, Susan K.; Smith, Lauren B.; Silva, Sandra; et al.

CS Neurogenetics Laboratory, Univ. of California San Francisco, San Francisco, CA, 94143, USA

SO Nat. Genet. (1996), 12(4), 436-441 CODEN: NGENEC; ISSN: 1061-4036

DT Journal

LA English

AB Manic-depressive illness, or bipolar disorder (BP), is characterized by episodes of elevated mood (mania) and depression1. We

designed a multistage study in the genetically isolated population of the Central Valley of Costa Rica2,3 to identify genes that promote susceptibility to severe BP (termed BPI), and screened the genome of two Costa Rican BPI pedigrees (McInnes et al., submitted). We considered

only

individuals who fulfilled very stringent diagnostic criteria for BPI to

be
affected. The strongest evidence for a BPI locus was obsd. in 18q22-q23.
We tested 16 addnl. markers in this region and seven yielded peak lod
scores over 1.0. These suggestive lod scores were obtained over a far
greater chromosomal length (about 40 cM) than in any other genome region.
This localization is supported by marker haplotypes shared by 23 of 26

- affected individuals studied. Addnl., marker allele frequencies over portions of this region are significantly different in the patient sample from those of the general Costa Rican population. Finally, we performed an anal. which made use of both the evidence for linkage and for assocn. in 18q23, and we obsd. significant lod scores for two markers in this region.
- L9 ANSWER 18 OF 21 CA COPYRIGHT 1999 ACS

AN 125:2629 CA

- TI Analysis of chromosome 18 DNA markers in multiplex
- pedigrees with manic depression

 AU Coon, Hilary: Hoff, M.; Holik, J.; Hadley, D.; Fang, N.; Reimherr, F.;
- Wender, P.; Byerley, William CS Medical School, University Utah, Salt Lake City, UT, 84132, USA
- SO Biol. Psychiatry (1996), 39(8), 689-696 CODEN: BIPCBF: ISSN: 0006-3223

DT Journal

LA English

- AB Six pedigrees segregating manic-depressive illness (MDI) were analyzed for linkage to 21 highly polymorphic microsatellite DNA markers on chromosome 18. These markers span almost the entire length of the chromosome, and gaps between markers are less than
 - cM. In particular, we analyzed several markers localizing to the pericentromeric region of chromosome 18 which generated lod scores suggestive of linkage in an independent study. Lod score anal. was performed and results were examd. by family. One region produced pos. lod scores, though at 18g23 and not in the pericentromeric region. We addml. used two nonparametric methods because the true mode
- of transmission of MDI is unknown; results were again somewhat suggestive
- for markers in the region of 18q23 but not in the pericentromeric region.
- L9 ANSWER 19 OF 21 CA COPYRIGHT 1999 ACS

AN 124:334471 CA

TI Linkage analysis of families with bipolar illness and

chromosome 18 markers

- AU <u>De bruyn</u>, An; Souery, Daniel; Mendelbaum, Karine; Mendlewicz, Julien; Van Broeckhoven, Christine
- CS Neurogenetics Laboratory, University Antwerp (UIA), Antwerpe, B-2610,
- SO Biol. Psychiatry (1996), 39(8), 679-688
- CODEN: BIPCBF; ISSN: 0006-3223

DT Journal LA English

AB Linkage of bipolar (BP) illness with chromosome

18 markers located at 18pl1 was recently reported. A possible role for chromosome 18 in the etiol. of BP illness was implicated previously by the finding in three unrelated patients of a

ring chromosome with breakpoints and deleted segments at 18pter-p11 and



18q23-qter. To test the potential importance of a gene defect on chromosome 18 in our material, we examd. linkage with chromosome 18 markers in two families with multiple patients with BP illness or BP spectrum disorders. Fourteen simple

tandem

repeat polymorphisms were used located in the chromosomal region 18p11 to 18q23 and sepd. by distances of approx. 10 cM on the genetic map. In one family linkage to chromosome 18 could not be excluded.

Linkage and segregation anal. in the family suggests that the 12-cM

region

between D18S51 and D18S61 located at 18q21.33-q23 may contain a candidate gene for BP illness.

L9 ANSWER 20 OF 21 CA COPYRIGHT 1999 ACS

AN 124:108441 CA

TI Evidence for linkage of bipolar disorder to chromosome 18 with a parent-of-origin effect

AU Stine, O. Colin; Xu, Jianfeng; Koskela, Rebecca; McMahon, Francis J.; Gschwend, Michele; Friddle, Carl; Clark, Chris D.; McInnis, Melvin G.; Simpson, Sylvia G.; et al.

CS School Medicine, Johns Hopkins University, Baltimore, USA

SO Am. J. Hum. Genet. (1995), 57(6), 1384-94 CODEN: AJHGAG; ISSN: 0002-9297

DT Journal

LA English

AB A susceptibility gene on chromosome 18 and a

parent-of-origin effect have been suggested for bipolar affective disorder (BPAD). We have studied 28 nuclear families selected for apparent unfilmear transmission of the BPAD phenotype, by using 31 polymorphic markers spanning chromosome 18. Evidence

for linkage was tested with affected-sib-pair and LOD score methods under two definitions of the affected phenotype. The affected-sib-pair

analvses

indicated excess allele sharing for makers on 18p within the region reported previously. The greatest sharing was at D18337: 64% in bipolar and recurrent unipolar (RUP) sib pairs (P = .0006). In addm., excess sharing of the paternally, but not maternally, transmitted alleles was obsd. at three markers on 18g: at D18941, 51 bipolar and RUP sib pairs were concordant for paternally transmitted alleles, and 21 pairs were discordant (P = .0004). The evidence for linkage to loci

both 18p and 18q was strongest in the 11 paternal pedigrees, i.e., those in which the father or one of the father's sibs is affected. In these pedigrees, the greatest allele sharing (81%; P = .00002) and the highest LOD score (3.51; THETA. = 0.0) were obsd. at D18841. Our results

provide

on

further support for linkage of BPAD to chromosome 18 and the first mol. evidence for a parent-of-origin effect operating in this disorder. The no. of loci involved, and their precise location, require further study.

L9 ANSWER 21 OF 21 CA COPYRIGHT 1999 ACS

AN 121:55150 CA

TI Chromosome 18 DNA markers and manic

-depressive illness: evidence for a susceptibility gene AU Berretthni, Wade H.; Ferraro, Thomas N.; Golddin, Lynn R.; Weeks, Daniel S:, Detera-Wadleigh, Sevilla; Nurnberger, John I., Jr.; Gershon, Elliot

S.
CS Dep. Psychiatry and Human Behavior, Thomas Jefferson Univ., Philadelphia, PA, 19107, USA

SO Proc. Natl. Acad. Sci. U. S. A. (1574), 91(13), 5918-21

CODEN: PNASA6; ISSN: 0027-8424

DT Journal

LA English

AB In the course of a systematic genomic survey, 22 manic

-depressive (bipolar) families were examd. for linkage to 11 chromosome 18 pericentromeric marker loci, under dominant and recessive models. Overall logarithm of odds score anal. for the pedigree series was not significant under either model, but several families yielded logarithm of odds scores consistent with linkage under dominant or recessive models. Affected sibling pair anal. of these data yielded evidence for linkage (P<0.001) at D18521. Affected pedigree member anal. also suggests linkage, with multilocus results for five loci giving P<0.0001 and P=0.0007 for weighting functions f(p)=1 and 1/.sqroot.p., resp., where p is the allele frequency. These results imply a susceptibility gene in the pericentromeric region of chromosome 18, with a complex mode of inheritance. Two plausible candidate genes, a ACTH receptor and the .alpha. subunit of a GTP binding protein, have been localized to this region.

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ANSWER 1 OF 7 BIOTECHDS COPYRIGHT 1999 DERWENT INFORMATION LTD
L10
AN
      99-04694 BIOTECHDS
TT
      New isolated fsh05 gene;
         associated with human bipolar affective disorder, useful for
         drug screening, diagnosis, therapy, mapping and DNA polymorphism
         identification of central nervous system disease, e.g. stroke
      Chen H; Freimer N B
AU
      Millennium-Pharm.; Univ.California
PΑ
LO
      Cambridge, MA, USA; Oakland, CA, USA.
      WO 9904825 4 Feb 1999
PΤ
      WO 98-US15183 22 Jul 1998
AΙ
PRAI
     US 97-898082 22 Jul 1997
DT
      Patent
LA
      English
08
      WPI: 99-142616 [12]
      ANSWER 2 OF 7 BIOTECHDS COPYRIGHT 1999 DERWENT INFORMATION LTD
AN
      99-02653 BIOTECHDS
TΙ
      New isolated human fsh05 gene;
         recombinant fsh05 gene, protein and antibody used to diagnose and
         treat neuropsychiatric disorder
      Chen H; Freimer N B
ΔH
PA
      Millennium-Pharm.; Univ.California
      Cambridge, MA, USA; Oakland, CA, USA. WO 9842362 1 Oct 1998
LO
PΤ
      WO 98-U$6208 27 Mar 1998
AT
      US 97-8/28010 27 Mar 1997
PRAI
DT
      Patent
LA
      English
OS
      WPI: 99-070062 [06]
L10
      ANSWER 3 OF 7 BIOTECHDS COPYRIGHT 1999 DERWENT INFORMATION LTD
AN
      99-02055 BIOTECHDS
      New isolated human fsh16 gene;
TΙ
         protein and antibody used for neuropsychiatric condition diagnosis,
         therapy and drug screening, and to identify fsh16 gene polymorphism
AU
      Chen H; Freimer N B
PA
      Millennium-Pharm.; Univ. California
LO
      Cambridge, MA, USA; Oakland, CA, USA.
      WO 9842726 1 Oct 1998
PT
      WO 98-USÉ210 27 Mar 1998
AΤ
PRAI
     US 97-828009 27 Mar 1997
DT
      Patent
      EngZish
T.A
      WPI: 99-045133 [04]
1.1.0
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AN
      99-00102 BIOTECHDS
TI
      New isolated human fsh15w6 gene;
         recombinant protein and encoding DNA for use in neuropsychiatric
         disease diagnosis, therapy and drug screening
      Chen H; Freimer N B
AU
      Millennium-Pharm.; Univ.California
PA
      Cambridge, MA, USA; Oakland, CA, USA.
LO
      WO 9842724 1 Oct 1998
PI
AΙ
      WO 98-US6211 27 Mar 1998
PRAI US 97-828007 27 Mar 1997
DT
      Patent
LA
      English
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WPI: 98-542273 [46]
      ANSWER 5 OF 7 BIOTECHDS COPYRIGHT 1999 DERWENT INFORMATION LTD
L10
AN
      99-00101 BIOTECHDS
TΙ
      New isolated human fsh22 gene;
         recombinant protein and encoding DNA for use in neuropsychiatric
         disease diagnosis, therapy and drug screening
ΔH
      Chen H; Freimer N B
      Millennium-Pharm.; Univ. California
PΑ
      Cambridge, MA, USA; Oakland, CA, USA.
      Wo 9842723 1 Oct 1998
PT
ZλT
      WO 98-US6209 27 Mar 1998
PRAI US 97-828008 27 Mar 1997
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OS
      WPI: 98-542272 [46]
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ΔN
      98-07280 BIOTECHDS
TOT
      New isolated IMP.18p myo-inositol-monophosphatase;
         human myo-inositol-monophosphatase gene-specific DNA primer
         construction, antibody and antisense DNA, used for manic
         -depressive illness susceptibility determination or therapy
ΔH
      Detera-Wadleigh S D; Gershon E S; Badner J A; Goldin L R; Berrettini W
н:
      Yoshikawa T; Sanders A R; Esterling L E
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      WPI: 98-272247 [24]
      ANSWER 7 OF 7 BIOTECHDS COPYRIGHT 1999 DERWENT INFORMATION LTD
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AN
TI
      Medical methods relating to bipolar mood disorder;
         genotype analysis for use in diagnosis
      Friemer N B; Leon P; Reus V I; Sandkuijl L A; Barondes S H
AU
PA
    Univ. California
LO
      Oakland, CA, USA.
     WO 9737043 9 Oct 1997
PI
    WO 97-US4904 27 Mar 19
ΆT
PRAI US 96-23438 23 Aug 1996; US 96-14498 29 Mar 1996
DT
      Patent
T,ZA
      English
0.5
      WPI: 97-535448 [49]
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116402 CHROMOSOME 213101 18 1804 CHROMOSOME (4A) (18) 12523 BIPOLAR 7876 MANIC

74 L8 AND (BIPOLAR OR MANIC) Lll

=> d 111 1-74

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C. Van Broeckhoven, Department of Molecular Genetics, Lab of Genetics, CS

University and Antwerp, Antwerp, Belgium

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United States CY

DT Journal; Conference Article

FS 022 Human Genetics

032 Psychiatry

A.T English SL English

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Van Broeckhoven C.; Raeymaekers P. Prof. C. Van Broeckhoven, Department of Molecular Genetics, Flandr. CS

Interuniv. Inst. Biotechnol., Universiteitsplein 1, B-2610 Antwerp,

Belgium. cvbroeck@uia.ac.be European Journal of Human Genetics, (1999) 7/4 (427-434). so

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CY United Kingdom

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ES 022 Human Genetics 032 Psychiatry

Τ.Σ English

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Grierson A.J.; Van Groenigen M.; Grootz N.P.B.; Lindblad K.; Hoovers AU J.M.N.; Schalling M.; De Belleroche J.; Baas F.

F. Baas, University of Amsterdam, Academic Medical Center, PO Box 22700, 1100DE Amsterdam, Netherlands. f.baas@amc.uva.nl

SO European Journal of Hyman Genetics, (1999) 7/1 (12-19).

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     ISSN: 1018-4813 CODEN: EJHGEU
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             Neurology and Neurosurgery
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     032
             Psychiatry
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     Nothen M.M.; Cichon S.; Rohleder H.; Hemmer S.; Franzek E.; Fritze J.;
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     Lichtermann D.; Maier W.; Craddock N.; Fimmers R.; Holler T.; Baur M.P.;
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     Dr. M.M. Nothen, Institute of Human Genetics, University of Bonn,
     Wilhelmstr 31, 53111 Bonn, Germany. noethen@humgen.uni-bonn.de
    Molecular Psychiatry, (1999) 4/1 (76-84).
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L11 ANSWER 5 OF 74 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V.
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     032
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             Drug Literature Index
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 - ΔN 1998018189 EMBASE
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     Lin J.; Bale S.J.
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6
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categories:
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AII
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CY
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L11 ANSWER 23 OF 74 EMBASE COPYRIGHT 1999 ELSEVIER SCI. B.V.
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TΙ
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ΑU
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AU
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CS
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DТ
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ΑU
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20892,
     United States
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 - FS 022 Human Genetics
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- TI Genes and psychosis: Old wine in new bottles?.
- AU Baron M.
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ь12 21 L8 AND (BIPOLAR OR MANIC)

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1998:86986 LIFESCI AN

Linkage analysis of candidate loci in families with recurrent major TΙ depression

7117 Balciuniene, J.; Yuan, Q.-P.; Engstroem, C.; Lindblad, K.; Nylander, P.O.:

Sundvall, M.; Schalling, M.; Pettersson, U.; Adolfsson, R.; Jazin, E.E.* Dept Medical Genetics, Box 589, Uppsala University, S-751 23 Uppsala, CS Sweden

MOL. PSYCHIATRY, (19980300) vol. 3, no. 2, pp. 162-168. SO

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An integrated physical map of 18p11.2: A susceptibility region for TT bipolar disorder

Esterling, L.E.; Matise, T.Cox; Sanders, A.R.; Yoshikawa, T.; Overhauser, J.; Gershon, E.S.; Moskowitz, M.T.; Detera-Wadleigh, S.D.

Bldg 10, Room 3N218, Clinical Neurogenetics Branch, National Institute of Mental Health, National Institutes of Health, 9000 Rockville Pike, Bethesda, MD, 20892-1274, USA

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DΨ Journal

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- AU Garcia, J.I.; Zalba, G.; Detera-Wadleigh, S.D.; De Miguel, C.* CS Depto. de Bioquimica, Univ. de Navarra, 31080 Pamplona, Spain
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patients with bipolar affective disorder from the Faeroe Islands

AH Ewald, H.; Nyegaard, M.; Wang, A.; Vang, M.; Mors, O.; Kruse, T.A. Inst. for Basic Psychiatric Res., Dep. Psychiatric Demography, CS

Psychiatric

Hosp., Aarhus, Denmark

197.

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Meeting Info.: 984 5018: 6th World Congress on Psychiatric Genetics (9845018). Bonn (Germany). 6-10 Oct 1998. International Society of Psychiatric Genetics.

- DТ Conference
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- ΑU McMahon, F.J.; Xu, Jianfeng; Stine, C.; Simpson, S.G.; DePaulo, J.R. Johns Hopkins, Baltimore, MD 21287-7381, USA
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- ΑU Sanders, A.R.; Yoshikawa, T.; Detera-Wadleigh, S.; Gershon, E.S.
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L13 ANSWER 6 OF 6 CONFSCI COPYRIGHT 1999 CSA
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            153 S MANIC AND DEPRESS? AND (18?)
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L4
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               4 S L5 NOT L2
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